Company, it was in fact reformulated locally from Prostin E_2 (dinoprostone vaginal suppository, 20 mg) and placed in a methylcellulose vehicle according to the method described by Gauger.[4] Because the reformulation process results in dehydration and isomerization of PGE_2 to inactive metabolites, the interpretation of study results must be viewed critically. We suggest that the lack of demonstrable clinical benefit with the use of endocervical PGE_2 in this investigation is likely due to an unknown but reduced dosage of PGE_2 in the extemporaneous formulation.

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REFERENCES

1. Gilson GJ, Izquierdo LA, Chatterjee MS, Curet LB, Qualls CR: Prevention of cesarean section—Does intracervical dinoprostone work? West J Med 1993; 159:149-152
2. Noah ML, DeCoster JM, Fraser TJ, Orr JD: Preinduction cervical softening with endocervical PGE_2 gel: A multicenter trial. Acta Obstet Gynecol Scand 1987; 66:3-7
3. Bernstein P: Prostaglandin E_2 gel for cervical ripening and labour induction: A multicentre placebo controlled trial. Can Med Assoc J 1991; 145:1249-1254
4. Gauger LJ: Extemporaneous preparation of a dinoprostone gel for cervical ripening. Am J Hosp Pharm 1983; 40:2195-2196

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Dr Gilson Responds

To the Editor: We appreciate Dr Noah's interest in our article and were similarly struck by the unexpected results of our study. Nevertheless, as detailed in our metanalysis, other prospective studies have arrived at similar conclusions. We took great care that the reformulated dinoprostone we used was not altered during preparation or storage. At the time our study was carried out, Prepidil Gel (Upjohn) was not available for clinical use, and so the 20-mg vaginal suppository, Prostin E_2 (Upjohn) was used.

As noted in our article, the procedure we followed consisted of manipulating the suppository by slicing it into thin sections and allowing it to soften at room temperature for 15 minutes. The softened slices were then geometrically mixed with the contents of a 56-gram tube of sterile hydroxyethylcellulose gel, K-Y Jelly (Johnson & Johnson), to yield a homogenous mixture. Homogeneity was assured by adding a small drop of 1% methylene blue to each slice to serve as a marker. Individual doses of the mixture (0.5 mg per 1.42 ml of gel) were drawn up into plastic syringes and stored at −20°C for as long as 35 days unexposed to light. This product was removed from the freezer approximately an hour before use to allow thawing.[1,2] In a related article, cocoa butter suppositories were also used as a vehicle for the dinoprostone.[3] During the melting process in a 37°C water bath, temperatures of the molten mixture greater than 33°C resulted in aberrations in the bioactivity of the drug. We therefore avoided this vehicle and liquefaction technique.

We conclude that the short and gentle reformulation process we used is unlikely to have resulted in a loss of bioactivity or bioavailability of the active substance. It must be stressed, however, that we conducted no chemical stability tests on these extemporaneously compounded products. The current high cost of the new commercially available gel may result in the continued clinical use of such extemporaneous formulations, and so Dr Noah's comments are well taken.

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REFERENCES

1. Gauger LJ: Extemporaneous preparation of a dinoprostone gel for cervical ripening. Am J Hosp Pharm 1983; 40:2195-2196
2. Curet LB, Gauger LJ: Cervical ripening with intravaginal prostaglandin E_2 gel. Int J Gynecol Obstet 1989; 28:221-228
3. Gauger LJ, Curet LB: Comparative efficacy of intravaginal prostaglandin E_2 in the gel and suppository forms for cervical ripening. DICP 1991; 25:456-460

More on the Crisis in Medical Education

To the Editor: I would like to make several comments regarding the editorial by Thomas Cesario, MD, in the August 1993 issue.

First I would like to commend Cesario's thinking through of the problem of increasing demands on physicians in academic centers and the effect that this has on medical student teaching. I do not, however, understand his conclusion that cost-effective medicine precludes "good clinical judgment, effective clinical decision making, and useful clinical algorithms." We must get away from the notion that every disease with similar symptoms has to be ruled out in every patient. The incidence of diseases seen in an academic center is not in any shape or form similar to that found in the community. Bayes's theorem is applicable. Not all tests are "good" tests. Common things are common. We must teach good, cost-effective medicine. "Good" and "cost-effective" can and should be two sides of the same coin.

My second observation is that Cesario's solutions of recruiting good community physicians to teach and rewarding them are certainly not novel. Family medicine has been doing this for years. In many medical schools family physicians from the community who like to teach are given academic appointments and receive stipends and other benefits for teaching medical students in their offices. Some receive no monetary reward but do it for the love and challenge of teaching and to repay society for some of the costs of their own medical education.

My point is not to toot the horn of family medicine but to say that those of us in the academic centers need to descend from the ivory towers of academe and learn from our colleagues in other specialties. It is expensive to keep reinventing the wheel. We should look to other specialties and copy and modify their educational strategies to fit our own specialties' needs. If internal medicine had done this years ago, that specialty would not need to try to maintain three sets of faculty—clinical, research, and instructional—in medical centers.

Because most patients are not treated in medical centers, it is necessary to use the whole community as the educational arena. Students learned this and demanded it long before academicians were willing to admit it. Let's do

what the patients need—train students to treat patients, not the rare diseases seen in medical centers.

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REFERENCE

1. Cesario TC: A crisis in medical education—One person's concern (Editorial). West J Med 1993; 159:212-213

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Dr Cesario Responds

TO THE EDITOR: Dr Taylor and I have no disagreement. My comment regarding clinical judgment and cost-effectiveness was made to indicate that I think they are mutually compatible. They have, in fact, similar implications merely viewed from two different perspectives—hence, opposite sides of the same coin.

Nor do I have any disagreement with the ability of family practitioners as excellent teachers and role models. I appreciate the many fine teachers who are practitioners of family medicine and am eager to include practicing physicians in our training programs because they constitute important role models for students.

I would, however, strongly disagree with the notion that internal medicine has been reinventing the wheel. We have sought to maintain standards of academic credibility and have done critical investigations when necessary to yield new knowledge on which clinical judgment or educational methods could be based.

It is not practical under the current system to suggest that many physicians can still competitively do clinical work, research, and instruction all at the same time when the competition for research dollars has become so fierce. The demands of the clinic are such now that considerable time is necessary to satisfy the expectations of practice groups and of patients. This is not to say that it is impossible for practitioners to do good research or to participate in outstanding projects. What is difficult, however, is for them to constantly maintain the funding and the support necessary to pursue the type of research that will yield essential new information to advance our understanding of the functions, the diseases, and the therapies needed to heal the human body.

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Capnocytophagal Pneumonia in a Healthy Man

TO THE EDITOR: *Capnocytophaga* species (DF-1) are facultatively anaerobic, slender gram-negative rods found as part of the oral flora. Serious capnocytophagal infections usually occur in immunocompromised patients.[1] We report a case of capnocytophagal pneumonia in an otherwise healthy young man.

Report of a Case

Malaise, sore throat, and nonproductive cough developed in a 25-year-old sailor. The patient's cough worsened, and severe paroxysms of coughing developed, followed by emesis and gasping respirations. Subsequently, he had shortness of breath at rest, fever to 38.9°C, and produced purulent sputum. On the third day of his illness, the patient was admitted to the sick bay of his ship with clinical signs of pneumonia, and empiric treatment with ceftriaxone, 1 gram every 12 hours, was initiated.

The patient was transferred to our facility four days after the onset of his illness. He appeared ill and had a temperature of 39.7°C. Bronchial breath sounds were present in the posterior basal lung fields. With the patient breathing room air, blood gas measurements showed an arterial pH of 7.48, a P_{O_2} of 50 mm of mercury, and a P_{CO_2} of 33 mm of mercury. A chest x-ray film showed diffuse interstitial and alveolar infiltrates with lower lobe predominance. Gram's stain of sputum revealed sheets of neutrophils and numerous slender, fusiform, gram-negative rods.

Therapy was initiated with the combination drug ticarcillin disodium and clavulanate potassium, 3.1 grams every four hours; tobramycin sulfate, 5 mg per kg of body weight per day; and erythromycin, 1 gram every six hours. Three sputum specimens obtained for culture during the first two days of hospital admission grew the *Capnocytophaga* species (DF-1). The *Capnocytophaga* organism was strongly β-lactamase-producing by Cefinase disc testing (BBL Cefinase, Becton Dickinson, Cockeysville, Maryland), and antibiotics were changed to the combination product ampicillin sodium and sulbactam sodium. The fever subsided over the next three days, and antibiotics were changed to an oral combination of amoxicillin and clavulanate potassium. His cough and hypoxemia resolved, and he was discharged on the fifth hospital day.

Discussion

Capnocytophaga species (DF-1) are unusual causes of infection in immunocompetent hosts. In a review of 16 cases of capnocytophagal infections in immunocompetent persons, most of the patients had serious underlying illnesses including malignancy, trauma, or surgical therapy, chronic renal insufficiency, and cholangitis.[1] Many of the patients were elderly, and multiple organisms were common. An oral source of infection was implicated in these patients.

We postulate that our patient initially had an upper respiratory tract infection, and then aspiration pneumonia developed as a complication of severe coughing spasms and posttussive emesis. Subsequently, ceftriaxone selected for the β-lactamase-producing *Capnocytophaga* species. The failure of a third-generation cephalosporin to eradicate β-lactamase-producing *Capnocytophaga ochracea* has been documented in a neutropenic patient.[2]